

TREATMENT OF PHONOLOGICAL ANOMIA IN APHASIA: SOME SUGGESTIONS FOR USERS OF SIGNED LANGUAGE*

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Abstract

Aphasiologists agree that with damage to the left hemisphere of the brain, both those who use vocal and those who use signed forms of communication are in jeopardy of developing aphasia, an impaired ability to produce and/or perceive language. Of the many symptoms that are associated with aphasia, anomia (a word retrieval impairment of language) is the most common, but while treatments for phonological anomia (a type of anomia caused by defective phonological processes) are available for users of spoken language, the same cannot be said for users of signed language. This paper aims to fill that gap in the literature by making some suggestions for the treatment of phonological anomia in signed language users. In spoken languages, phonological word finding deficits can be remediated by phonemic cueing tasks in which the initial phonemes of target words are provided as cues to phonologically prime targets and facilitate their retrieval. In this paper I argue that by simply substituting phonemes as cues for cheremes (the signed language equivalent of phonemes), we could adapt the phonemic cueing approach to treatment of phonological anomia in spoken languages to make it suitable for use with signed language users.

1. Introduction

The study of signed language linguistics has long established that signed languages are natural human languages in the way that spoken ones are (Stokoe 1960, Klima & Bellugi 1979, Senghas et al. 2004) and that while the two modalities differ in their methods of production and perception, their underlying linguistic structures are the same (Sandler & Lillo-Martin 2006). We also know from brain imaging studies that the left cerebral hemisphere of the brain is responsible for processing all modalities of language whether those be spoken or otherwise (Bavelier et al. 1998, Hickok et al. 2002), so one would expect that when deaf signers endure neurological damage to their left cerebral hemisphere, the consequences for language for them would be very much the same for them as they are for hearing speakers.

This expectation seems to be supported: aphasiologists agree that with left cerebral hemisphere damage those who use vocal forms of communication are in jeopardy of developing aphasia, an impaired ability to produce and/or perceive language (Naeser & Hayward 1978, Kertesz et al. 1979, Damasio 1989, Damasio 1992, Fitzgerald 1996, Corina 1998, Kolb & Wishaw 2003, Kearns 2005, Keller et al. 2009) and a number of studies have confirmed this to be the case for signed language users too (Leischner 1943, Tureen et al. 1951, Douglas & Richardson 1959, Poizner et al. 1987, Corina et al. 1992, Hickok et al. 1998, Corina et al. 1999, Hickok et al. 2011). One of the symptoms most commonly shared among people with aphasia is anomia, a word retrieval impairment of language (Goodglass & Wingfield 1997). Two different types of anomia have been described in the aphasiology literature: semantic anomia, which is caused by defective semantic processes and phonological anomia, which is caused by problems with the phonological processing system (Nettleton & Lesser 1991, Raymer et al.

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1993, Hillis & Caramazza 1994, Boyle & Coelho 1995, Miceli et al. 1996, Nickels & Best 1996, Drew & Thompson 1999, Kiran & Thompson 2003), but while treatment for phonological anomia is available for users of spoken language, the same cannot be said for users of signed language. This paper aims to fill that gap in the literature by making some suggestions for the treatment of phonological anomia in signed language aphasia from the perspective of theoretical linguistics.

The body of discussion is categorised into five main parts (Sections 2, 3, 4, 5 and 6). The first part (Section 2), presents an introductory account of the major characteristics or symptoms of aphasia observed in spoken and signed languages; while Section 2.1 addresses aphasia in spoken languages, Section 2.2 addresses it in signed ones. Section 3 hones in on anomia, an impairment of language that is common in aphasia and characterised by an inability to access words in and retrieve them from the mental lexicon. It describes the differences between semantic anomia and phonological anomia, and points out the need for treatment for phonological anomia in signed languages. Sections 4 and 5 give an overview of the theory underpinning phonologically targeted treatment for phonological anomia. While Section 4 concerns itself with how the mental lexicon is organised and how words are accessed in it, Section 5 describes how phonological priming, an implicit memory effect that is triggered in phonological intervention for phonological anomia, can influence lexical access. Section 6 describes a particular type of treatment for phonological anomia for hearing speakers with aphasia (phonemic cueing treatment) before making some suggestions for its adaptation for use with signed language users. Section 7 summarises and concludes.

2. Aphasia

2.1. Spoken language aphasia

Aphasia is an acquired disorder of language that presents as a result of damage to the brain areas responsible for the production and/or comprehension of language and its components (Helm-Estabrooks et al. 2014). Depending on the site or sites of damage and the relative impairment or sparing of the language components, different subtypes of aphasia can emerge (Helm-Estabrooks et al. 2014). There are two major subtypes of aphasia: Broca's aphasia and Wernicke's aphasia (which are caused by damage to the lateral frontal, suprasylvian, pre-Rolandic area or the adjacent subcortical periventricular white matter, and the posterior third of the superior temporal gyrus, respectively). Broca's aphasia is otherwise known as expressive aphasia and is so named because it is characterised by impaired language production with unimpaired language comprehension (Kerterz 1982). Wernicke's aphasia, on the other hand, (which can also go by the name of receptive aphasia) causes problems of a similar vein but in the opposite direction: while a Broca's patient experiences problems with language production but not its comprehension, a Wernicke's patient has them with its comprehension, but not its production (Albert et al. 1981). Other more minor subtypes of aphasia include conduction aphasia, transcortical motor aphasia, transcortical sensory aphasia and global aphasia (Helm-Estabrooks et al. 2014), and with all of the different subtypes, different symptoms are associated. Some level of anomia, meanwhile, is seen in all of the aphasias (Manasco 2014).

2.2. Signed language aphasia

It has long been established that the signed languages of deaf communities are fully-fledged human languages in the way that the spoken languages of hearing communities are (Stokoe 1960, Klima & Bellugi 1979, Senghas et al. 2004) in that there are a number of autonomous mutually unintelligible systems of communication used in deaf communities around the world (Woll et al. 2001, MacSweeney et al. 2008). It is also generally accepted by linguists that while

spoken languages and signed languages differ in their methods of production and perception (in that spoken languages are produced by the mouth and perceived by the ears whereas signed languages are produced by the hands and perceived by the eyes), their underlying linguistic structures are the same (Sandler & Lillo-Martin 2006). This is primarily because key linguistic features (or language universals) in the theoretical domains of phonology, syntax and semantics identified in spoken languages have been identified in signed languages as well, but similarities observed between the neural bases of spoken and signed languages have also been taken to reflect that all linguistic functioning is underpinned by the same system. Neuroimaging studies have found evidence to suggest that like spoken languages, signed languages are processed by the left hemisphere of the brain (see Hickok et al. 1998), that both speech and sign production is processed by the left inferior frontal gyrus (Braun et al. 2001, Corina et al. 2003, Emmorey et al. 2003, Emmorey et al. 2007) while speech and sign comprehension rely on the left superior temporal gyrus and sulcus for their processing (Neville et al. 1998, MacSweeney et al. 2002, Newman et al. 2002, Meyer & Damien 2007, Corina et al. 2007, Capek et al. 2008).

If signed language processing like spoken language processing is left dominant, this should be reflected by the patterns of impairment that follow brain damage (Marshall et al. 2004), should it not? That is, aphasia in signed languages should follow left- but not right-hemisphere damage in the way that it does in spoken ones. A number of lesion studies in signers have confirmed that this is indeed the case (e.g. Poizner et al. 1987, Corina 1998, Marshall et al. 2004 and Atkinson et al. 2005) and there is even evidence to suggest that the symptoms found in signed language impairments are consistent with those found in spoken language impairments.

In 1987, for example, Poizner et al. reported six cases of unilateral stroke in deaf users of American Sign Language (ASL). While those with left hemisphere damage presented with language problems in their case studies, those with right hemisphere damage did not. What's more they found that damage to the left frontal regions resulted in production difficulties (as we see in spoken language Broca's patients) while damage to the left temporal lobe caused problems with comprehension (just like in spoken language Wernicke's aphasia). These findings have been supported by a number of other signed language researchers (see Corina et al. 1992 for just one representative example).

People with signed language aphasia have also been said to experience the word retrieval difficulties that people with spoken language aphasia are all too familiar with. Marshall et al. (2004), for instance, described a case of aphasia in a user of British Sign Language (BSL) called Charles which arose as a result of a left cerebrovascular accident. A computer tomography (CT) scan revealed damage to the left posterior frontal and parietal lobes extending into the corona radiata and temporal lobe, and as one would predict, Charles' resultant language problems were with production. He struggled to retrieve the correct forms of signs and frequently was unable to access signs at all, but in contrast to his production problems, his comprehension of signs was unaffected. In an informal comprehension test Charles scored 10/10, indicating that he had a Broca's-like aphasia.

Convinced that Charles had anomia as a symptom of his aphasia, Marshall et al. (2004) administered a number of assessments to see whether their predictions could be proven. In a picture naming task which required him to name pictures on confrontation Charles scored only 27/37, leading Marshall et al. (2004) to conclude that he did indeed have a problem with the retrieval of words. In 1982 Chiarello et al. witnessed a similar situation in their investigation of a prelingually deaf woman who had suffered a left parietal infarct involving the left supramarginal and angular gyri. She too developed a sign retrieval impairment that mirrored the word retrieval impairments of oral aphasic patients.

Now although there is evidence to suggest that anomia is common to both the spoken and the signed modalities of language, there has been little to no research into its treatment in

signed language aphasia. In this paper I aim to inform the neglected area of research by suggesting that we adapt current treatment for phonological anomia in spoken languages for use with signed language users, but before any suggestions can be made, however, we must first look at anomia in more detail.

3. Anomia

As we observed above, anomia is an impaired ability at accessing words in and retrieving them from the mental lexicon (Goodglass & Wingfield 1997) which can range from a mild difficulty in producing desired words during conversational discourse to a virtual inability to produce them under any conditions at all (Helm-Estabrooks et al. 2014). This section will provide a more thorough, comprehensive account of anomia. It will begin with an overview of its causes before describing its symptoms, diagnosis and treatment.

3.1. Its causes

As a symptom of aphasia, anomia is thought to result from damage to regions in the left hemisphere of the brain (Woollams et al. 2008). This damage can either be traumatic or acquired (Damasio 1992) with causes of traumatic brain injury including the likes of falls, accidents or violence involving a blow to the head and causes of acquired brain injury poisoning, infection, strangulation, choking, drowning, stroke, heart attacks, brain tumours, aneurysms and even neurodegenerative diseases (such as Parkinson's disease, Alzheimer's disease and Huntington's disease) (Budd et al. 2010). It was initially thought that damage to Broca's area (the speech production centre of the brain) or Wernicke's area (the speech comprehension centre) could be held responsible for the onset of anomia (see Fridriksson et al. 2009 and Hamilton et al. 2010, for example), but more recent studies have suggested that anomia is caused by damage to the arcuate fasciculus (the nerve tract that connects the caudal temporal cortex and inferior parietal cortex to locations in the frontal lobe) more specifically (Catani & Thiebaut de Schotten 2008, Carlson 2012).

3.2. Its symptoms

The most accurate way of determining whether anomia has developed in an individual is through tests, though there are a number of symptoms that can point to its presence. Unsuccessful attempts at word retrieval tend to result in speech errors that resemble the target in meaning (semantic speech errors) or in sound (phonological speech errors), which are therefore taken to be symptomatic of anomia.

Paraphasias are a particular type of speech error that are commonly observed in people with aphasia. These can be divided into two broad classes: semantic paraphasias in which an irretrievable target word is substituted with a word that is semantically related to it such as *refraction* for *reflection* (which both are to do with the dispersion of light) (Damasio 1992, Fitzgerald 1996, Marshall et al. 1998) and phonological paraphasias which substitute irretrievable target words with words or nonwords that resemble them phonologically like *viscosity* ([vɪskɒsɪti]) for *velocity* ([vɪlɒsɪti]) (a real word, formal error) or *chromosone* ([krəʊməsəʊn]) for *chromosome* ([krəʊməsəʊm]) (a nonword phonemic error) (Brookshire 1997).

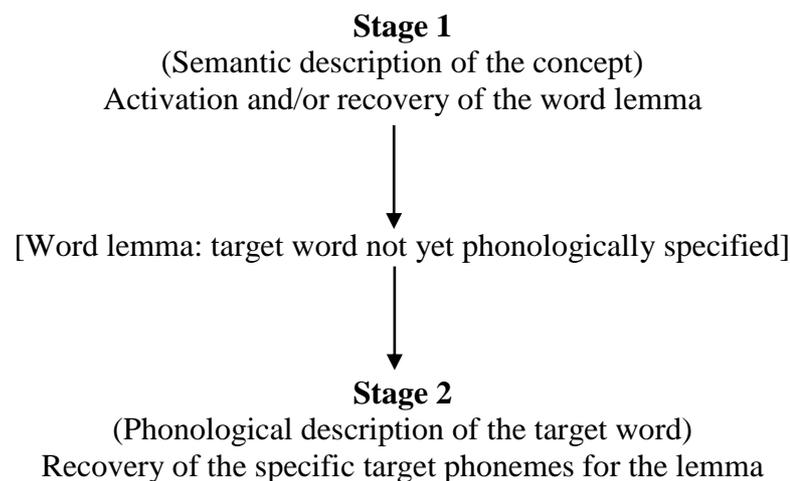
Semantic paraphasias suggest that while phonological information about a target word can be successfully retrieved by the patient, not all of its semantic information can be. Phonological paraphasias, on the other hand, in particular phonemic errors in which only partial phonological information about a target is retrieved and the rest is deformed by the likes of

addition, deletion, substitution or transposition (Lecours & Lhermitte 1969, Blumstein 1973) suggest that while semantic information about a target is available for retrieval, there are problems with a patient's access to the sounds constituting its spoken form.

3.3. Its diagnosis

In 1979 Benson drew a distinction between two types of anomia: semantic anomia and phonological anomia (Devinsky & D'Esposito 2004) and one way that researchers have tried to understand the difference between the two and how to diagnose them is by studying word retrieval errors like the paraphasias described above. An approach that many aphasiologists find useful for diagnosis of the anomias is one that characterises a patient's word retrieval problems with reference to a cognitive neuropsychological model of the word retrieval process outlining the different information processing stages involved in it (Ellis et al. 1992) as exemplified in Figure 1. Armed with a model like this, aphasiologists are able to relate the speech errors an aphasic patient produces to breakdown of one or more of the model's components (and ergo of one or more of a patient's cognitive operations):

Figure 1. A two-stage model of word retrieval (adapted from Helm-Estabrooks et al. 2014: 232)



Aphasiologists generally agree that word retrieval begins with the recovery of semantic information about a target word and ends with the recovery of its phonological information (Allport 1985), so two-stage models of word retrieval like the one in Figure 1 are helpful in that they draw a distinction between those two stages of the word retrieval process: a first stage in which an object is perceived and comprehended and a second stage in which the name for the object is retrieved (Helm-Estabrooks et al. 2014). It is thought that stages 1 and 2 of the model constitute separable cognitive components (the semantic system and the phonological system) that can be selectively impaired giving rise to two different types of anomia (semantic anomia and phonological anomia, respectively). It is thought that semantic paraphasias are caused by defective semantic processes and are therefore symptomatic of semantic anomia, while phonological paraphasias (caused by problems with the phonological processing system) are a symptom of phonological anomia, that failure to retrieve enough semantic information about a target to activate its appropriate word lemma at stage 1 leads to semantic speech errors, and failure to adequately activate stage 2's phonological description of a target causes phonological errors (Helm-Estabrooks et al. 2014).

Now although we can make a preliminary diagnosis of a patient's anomia by looking at the types of error they typically produce, all provisional diagnoses must be confirmed by a proper evaluation. A patient's comprehension of the words he or she is unable to retrieve might be evaluated using the Pyramids and Palm Trees test (Howard & Orchard-Lisle 1984), for example. Object recognition and comprehension can be assessed using the all picture version of the test which requires a patient to indicate which of two pictured objects belongs with a third pictured object while recognition and comprehension of their names can be assessed by asking a patient which of two pictured objects matches the name of a third object.

The Psycholinguistic Assessments of Language Processing in Aphasia (PALPA) Battery (Kay et al. 1992) can also be used to test for semantic word retrieval impairments. Comprehension of object names is assessed with a 40-item test in which a patient must indicate which of five pictures (including the target word itself, two semantic distractors, a visual distractor that is related in appearance but not in function to the target word and a word that is related in function to the visual distractor but not in its appearance) matches a heard word. Patients with semantic impairments will tend to make errors involving the semantic distractors, but if there is no obvious pattern in a patient's speech errors, their deficit will probably not be semantic in nature Kay et al. (1992) claim.

The possibility of a phonological impairment might then be explored using a set of pictures that was published by Snodgrass and Vanderwart in 1980. Patients who show an effect of word frequency when matching words to these pictures are often described as having phonological anomia, as are patients who have problems with the articulation of the longer words but not the shorter ones or who can demonstrate their understanding of the words but are unable to produce them (Ellis et al. 1992).

3.4. Its treatment

A number of different approaches have been shown to be effective in the remediation of word retrieval impairments in aphasia (Howard et al. 1985, Pring et al. 1990, Hillis & Caramazza 1994, Fick et al. 2002, Hickin et al. 2002, Raymer & Ellsworth 2002). Some aphasiologists argue that treatment should be targeted to the damaged level of language processing, that word retrieval difficulties caused by impaired word meaning respond best to semantically targeted treatments while phonologically targeted treatments are best used to treat word retrieval difficulties caused by damage to the phonological processing system (Nettleton & Lesser 1991, Miceli et al. 1996). Semantically targeted tasks include the likes of matching words to pictures, answering yes/no questions about pictures and picture categorisation (Marshall et al. 1990, Davis & Pring 1991, Byng 1995, Nickels & Best 1996) and phonologically targeted tasks phonemic cueing (whereby the word-initial phonemes of target words are provided to patients for processing) (Crofts et al. 2004).

While it has indeed been demonstrated that semantically targeted tasks are effective in the treatment of semantic anomia (e.g. Marshall et al. 1990, Pring et al. 1990, Nickels & Best 1996) there is also evidence to suggest that they can improve word retrieval for those who do not have an impairment at the semantic level (Nickels & Best 1996). This is also true for phonologically targeted tasks. It was also originally thought that phonological interventions for anomia should be used exclusively for those with phonological impairments (Nettleton & Lesser 1991, Hillis & Caramazza 1994, Miceli et al. 1996) but they have in actual fact been found to be successful at treating word retrieval impairments at the semantic level as well (Raymer et al. 1993, Nickels & Best 1996). The success of both types of treatment at improving word retrieval in each of the anomias could be attributed to the fact that they are not actually entirely different from one another after all, however. Howard (2000) points out that in semantically targeted treatments, the spoken form of a target word is provided evoking

phonological processing and in phonologically targeted treatments, a picture of a target is presented to the patient evoking semantic processing so in each of the treatments, both semantic and phonological information is available to the patient meaning they have their effects in the exact same way: by strengthening the connections between word meaning and word form.

Since Howard's (2000) observation predicts equivalent effects for individuals with semantic and phonological anomia with each of the different types of treatment, it may well be the case that adapting phonologically targeted treatments for use with signed language users could have consequences for individuals with semantic anomia as well as those with phonological anomia, but given that few other studies have examined the issue I think it would be presumptuous to make such a claim at such an early stage. In this paper, I will be making suggestions for the treatment of phonological anomia in signed languages only and my proposal is as follows. If it is true that the same phonological system that underlies spoken languages underlies signed ones, phonological processing should occur in each modality in the same way, meaning treatments that target phonological processing to improve word retrieval in hearing people with phonological anomia could have equivalent effects for deaf phonological anomics provided they were made suitable for their methods of production and perception. But before we begin to think too deeply about the ways in which word retrieval may be influenced, we must first explore how words are stored in the mental lexicon, how they are organised with respect to one another and how they can be accessed.

4. The mental lexicon

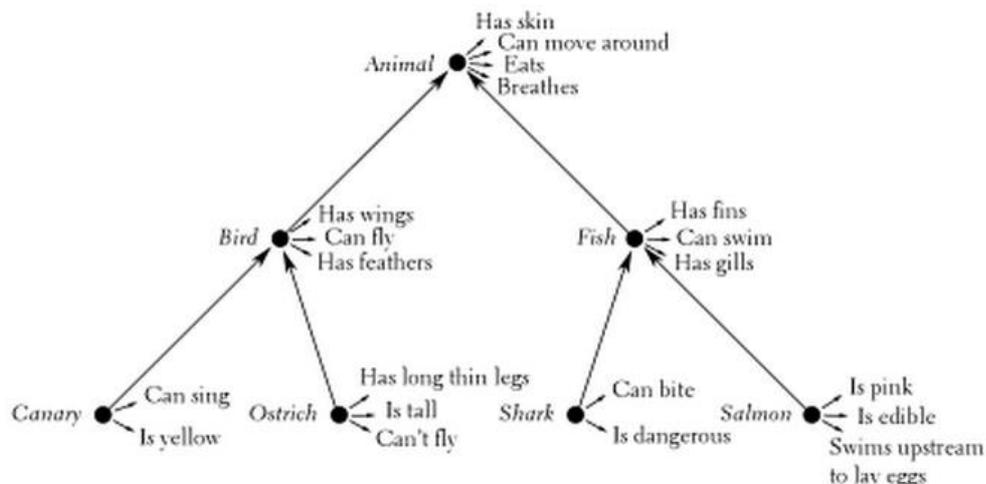
Psycholinguists argue that the underlying representations of words (and thus our knowledge of them) are stored in permanent memory in a mental lexicon (Collins & Quillian 1969, Caron 1989) from which they must be accessed. The study of lexical access sets out to answer two types of question: the first concerns how units are represented and organised while the second bears on the processes involved in accessing items in the lexicon. This section begins by addressing the first question before addressing the second.

4.1. Organisation of the mental lexicon

4.1.1. Hierarchical network models

Collins & Quillian's (1969) hierarchical network model was the first systematic model of semantic memory. It suggested that semantic memory is organised into a series of hierarchical networks consisting of nodes and their properties where nodes represent concepts such as *animal*, *bird* and *canary* and properties, rather self explanatorily, the characteristics of those concepts. The model is arranged with broader concepts on the higher levels and narrower concepts on the lower levels as exemplified in Figure 2:

Figure 2. A hierarchical network model of semantic information related to animals (Collins & Quillian 1969: 241)

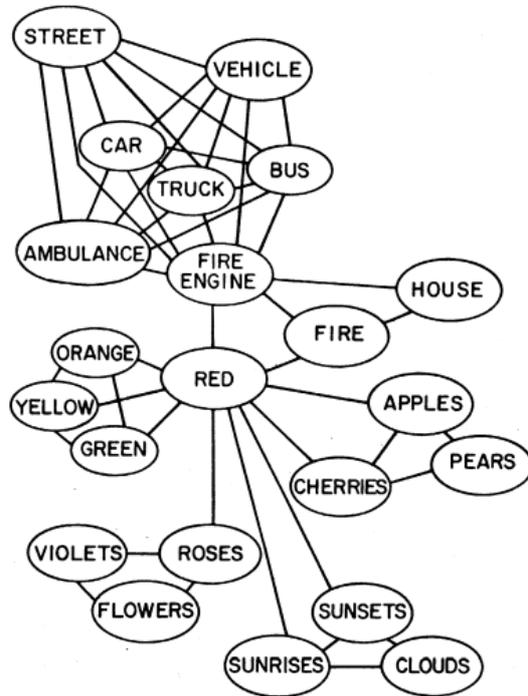


In Figure 2, the concept ANIMAL dominates the concepts BIRD and FISH as both birds and fish are animals. Likewise, CANARY and OSTRICH are dominated by BIRD as they are both types of bird and SHARK and SALMON by FISH as they are both types of fish, but although this sort of organisation in which broader categories are organised higher in the hierarchy than narrower categories seems to be logical, it also has its problems. Collins & Quillian's (1969) hierarchical network model assumes that all items on a given level of a hierarchy are more or less equal. Since SHARK and SALMON are both subordinates of fish and are organised on the same level as each other, they should, in theory, take the same amount of time to access in the mental lexicon. In actual fact, they do not, and this generally seems to be the case (Carroll 2008). Smith et al. (1974) found that items more typical of a given subordinate take less time to access than atypical items. For example, in everyday conversation, the word *salmon* is more likely to come up than the word *shark* is as it is a foodstuff. Being a higher frequency word its concept is accessed in the mental lexicon more quickly (Smith et al. 1974) and this 'typicality effect' suggests that a hierarchical network might not necessarily be the best way to model the mental lexicon after all.

4.1.2. Spreading activation models

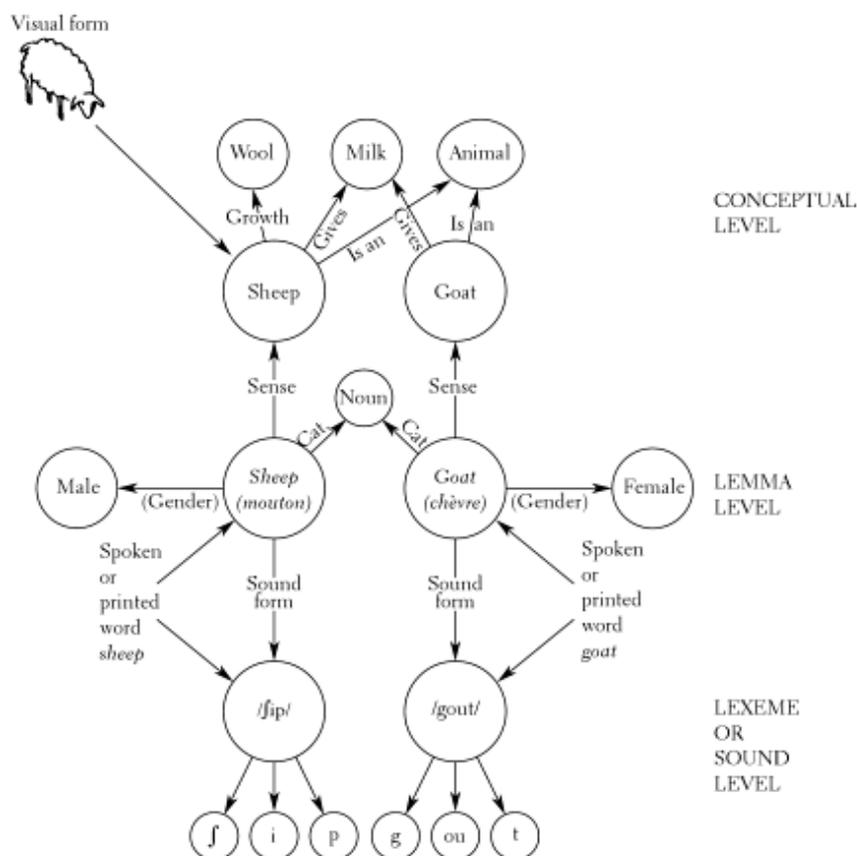
A second class of models known as spreading activation models retain Collins & Quillian's (1969) idea of a network but discard their hierarchical assumption. Collins & Loftus (1975) propose that the organisation of words is more like a web of interconnecting nodes than a hierarchy of them. They argue that retrieval occurs by a process of spreading activation which begins at a single node and spreads to its neighbouring nodes in the network attenuating over distance, ensuring that closely related concepts are more likely to be activated than distant concepts are as in Figure 3:

Figure 3. A schematic representation of a spreading activation model of semantic knowledge (Collins & Loftus 1975: 412)



According to Collins and Loftus' (1975) model, if the concept CAR was activated, the STREET, VEHICLE, BUS, TRUCK, AMBULANCE and FIRE ENGINE concepts are more likely to be activated than the SUNSETS, SUNRISES and CLOUDS concepts. This makes sense given that the first group of concepts are more similar in meaning to the concept CAR than the second group of concepts, but although Collins & Loftus' (1975) model is a step forward from Collins and Quillians' (1969) model, it too has its limitations. Very little information is paid to the phonological and syntactic aspects of words so in a sense it is more a model of concepts than a model of words.

A more recent spreading activation model that incorporates lexical as well as conceptual aspects was established by Bock & Levelt (1994):

Figure 4. Bock & Levelt's (1994) spreading activation model (Bock & Levelt 1994: 951)

Bock & Levelt (1994) assume that our knowledge of words exists at three different levels: a conceptual level, a lemma level and a lexeme level where the conceptual level is comprised of concepts, the lemma level of the syntactic aspects of word knowledge and the lexeme level a word's phonological properties (Levelt 1989). Spreading activation models like this one which incorporate conceptual, syntactic and phonological knowledge are thought to offer the most realistic picture of the mental lexicon (Posner & Snyder 1975, Neely 1977, 1991, Marcel 1983), but if this is how words are organised in the mental lexicon, how are they then accessed in it? This question is addressed in Section 4.2.

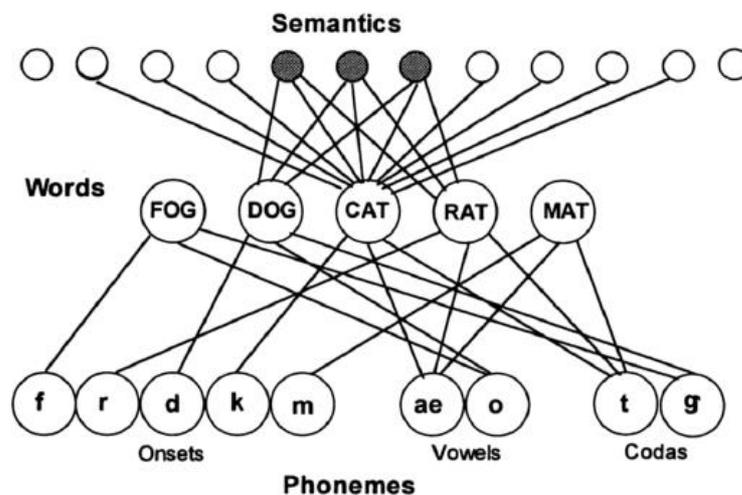
4.2. Lexical access

Theories of lexical access generally agree that word retrieval occurs in two stages: a first stage known as lemma access (which is the mapping from a conceptual representation to a lemma (an abstract conceptual form of a word that has been mentally selected for utterance in the early stages of speech production but has not yet assumed its phonological form)) and a second stage called phonological access (the mapping from the lemma to its phonological representation) (Fromkin 1971, Garrett 1975, Potter & Faulconer 1975, Fay & Cutler 1977, Kempen & Huijbers 1983, Dell 1986, Butterworth 1989, Glaser & Glaser 1989, Levelt 1989, La Heij 1990, Levelt et al. 1991, Roelofs 1992, Bock & Levelt 1994). The relationship between lemma selection and phonological encoding, however, has been subject to serious debate. While some models of lexical access assume that lemma selection is completed before the activation of any phonological information (see Levelt 1989 and Roelofs 1997, for example), other researchers have found indications that this may not be so (e.g. Peterson & Savoy 1998, Cutting & Ferreira

1999). In light of that newfound evidence, Dell et al. (1997) went on to develop a new model of lexical access which preserved earlier models' distinctions between lemma selection and phonological encoding but denied that those stages were modular.

For Dell et al. (1997), lexical knowledge is embedded in a network of three layers: a semantic layer, a word (or lemma) layer and a phonological layer as in Figure 5 where each unit in the word layer is connected bidirectionally to their semantic and phonological features allowing activation to spread in a top-down fashion from semantic units to word units to phonemic units and bottom-up from phonemic to word to semantic units. Being nonmodular, Dell et al.'s (1997) later stages may begin processing before the earlier ones have finished (cascading) and processing at the later levels may influence that at earlier ones (feedback):

Figure 5. An illustration of a lexical network in the two-step lexical access model of word retrieval (Dell et al. 1997: 805)



Dell et al. (1997) have actually been known to use their model to explain the error patterns one might observe in aphasic people with anomia. They argue that two steps are involved in the word retrieval process, lemma selection and phonological encoding, and that breakdowns at each can give rise to the speech errors we see in anomia.

In a scenario where a picture of a cat is presented to a person, they say, visual processes outside of the model will immediately identify that the concept illustrated by the picture is a cat and activate the semantic features of that concept. This activation then spreads through bidirectional excitatory connections for a fixed number of time steps according to a linear activation function until all three network levels are active, and in addition to the target word unit CAT, semantic neighbours such as DOG can become activated through shared semantic nodes, as can units such as MAT, CAT and CAN which, as phonological neighbours, can receive cascading activation from phonemes shared with the target. This first stage of the word retrieval process concludes with the most highly activated word being selected for retrieval, but this process is not always as straightforward as one might think, they point out. A semantic or phonological neighbour of the target word may well be selected if their activation levels exceed that of the target. This is an all too common occurrence in people with anomia.

This ties in with an explanation of anomia with the transmission deficit (TD) model in terms of the strength of the network connections that transmit activation to the representations involved in the retrieval of words (MacKay & Burke 1990, Burke et al. 1991). According to MacKay & Burke (1990) and Burke et al. (1991), semantic anomia occurs when the strength of connections among semantic representations become too weak through damage to transmit enough activation to the target word's semantic representation in the mental lexicon for it to be

retrieved, while damage to connections among phonological representations can result in phonological anomia by making them too weak to transmit sufficient activation to target words' phonological representations for their retrieval.

Phonological errors can also occur in the second stage of the word retrieval process, Dell et al. (1997) argue. In this stage, a chosen word is activated and activation spreads for another fixed number of time steps. As is the case for lemma selection, activation spreads both upwards and downwards during the phonological encoding process meaning that nodes other than the ones directly connected to CAT can be activated. The most highly activated phonemes are then selected for retrieval and errors occur when one or more phonemes are more active than those of the target word. This can result in real word phonological errors such as *mat*, *sat*, and *can* for *cat* and even nonword phonological errors such as *lat*.

Some variables have been said to influence how easily words are accessed in the mental lexicon (Carroll 2008). Among these are lexical factors such as typicality and frequency but perhaps the biggest influence on lexical access is a contextual factor known as priming. It is widely agreed that the processing of lexical items is affected by their preceding context; that people will tend to process a word more quickly and accurately when they have previously been exposed to a similar word (Goldinger et al. 1989, Marslen-Wilson & Zwitserlood 1989, Slowiaczek & Hamburger 1992). Word retrieval experiments for example have shown that people will tend to retrieve a word like *boy* more quickly if they have previously been exposed to a word like *girl* as opposed to something unrelated due to their similarity in meaning (semantic priming) and a word like *orgasm* more quickly if they have been previously exposed to a word like *organism* than something unrelated due to their similarity in sound (phonological priming) (McDonough & Trofimovich 2008).

In fact, it is precisely because phonological priming is known to affect lexical access that phonemic cueing tasks (which make use of it) are used in the treatment of phonological anomia, but how exactly does phonological priming occur and how can it improve word retrieval in phonological anomics, one might wonder? Sections 5 and 6 address those very questions.

5. Phonological priming and its influence on lexical access

5.1. Phonological priming in spoken languages

Phonological priming of spoken words refers to the improved recognition or retrieval of targets when they are preceded by primes that resemble them phonologically by sharing one or more of their constituent phonemes (James & Burke 2000). In phonological priming experiments, participants are typically presented with two words (a prime and a target) and asked to perform a task on the second to determine the effect hearing a prime has on the processing of a target (Pitt & Shoaf 2002).

In 1987, Slowiaczek et al. reported the results of three phonological priming experiments. In each of their three experiments, subjects were tasked with accurately identifying the target words before and after the provision of a prime. In the first experiment, the primes and targets were all real words and their shared phonemes appeared word-initially. Facilitatory effects were found for all of the primes. In the second experiment, nonwords were used as primes and similar effects were observed. Finally, in the third experiment, real word primes and targets were used once more, but rather than them being in word-initial positions, the shared phonemes were in word-final positions. Facilitatory effects were observed in the third experiment as well.

Slowiaczek et al. (1987) explained their results in terms of McClelland & Rumelhart's (1981) interactive-activation model of word recognition which like Dell et al.'s (1997) two-stage model of word retrieval assumes that phonemes and words exist on separate levels of

representation with excitatory activation passing between them. They argued that when a phonological prime is activated in the lexicon, the nodes for its phonemes are too and spread activation upwards through phoneme-to-word links to all other words that share those phonemes. These words remain partially active for a brief period, they claimed, allowing successful retrieval of the target word to be accomplished more easily than it would have been had a number of candidates for it to be selected from not been activated.

The results of other studies in the phonological priming literature would suggest, however, that effects largely depend on the extent to which primes' and targets' segments overlap. When primes overlap with targets by a small number of segments as was the case in Slowiaczek et al.'s (1987) study, facilitatory effects tend to be reported (e.g. Goldinger et al. 1992, Slowiaczek & Hamburger 1992, Hamburger & Slowiaczek 1996, 1999, Goldinger 1999, Spinelli et al. 2001) but when too many segments overlap inhibition of processing can be observed (Slowiaczek & Hamburger 1992, Hamburger & Slowiaczek 1996, 1999, Dufour & Peereman 2003).

Slowiaczek & Hamburger (1992), for example, conducted a priming experiment on targets preceded by primes that had no phonemes in common and therefore did not bear any phonological similarity to each other (e.g. clump (prime) and green (target)), primes that shared one phoneme with the target (e.g. goals (prime) and green (target)), primes that shared two phonemes (e.g. grope (prime) and green (target)) and primes that shared three (e.g. grief (prime) and green (target)) (Dufour & Peereman 2003). They found that response times were faster when primes shared one phoneme with the target than when there was no phonological similarity, but that the facilitatory effect decreased as the degree of phonemic overlap increased. This pattern of results led the researchers to conclude that too much similarity between a prime and a target can cause competition between the two during the word retrieval process and inhibit the target word's retrieval (Dufour & Peereman 2003). They argued that if there is a large phonemic overlap between a prime and a target, when the target is processed its prime is easily reactivated and acts as a strong competitor for retrieval, slowing down (and occasionally preventing) retrieval of the target word. When there is only a single phoneme overlap, however, the prime is not as easily reactivated and so does not interfere with the processing of the target (Dufour & Peereman 2003).

This can explain why priming with word-initial phonemes of targets in phonemic cueing treatments for phonological anomia can improve word retrieval but are not known to inhibit it. Because the primes used in phonemic cueing tasks are phonemes, not words, they cannot act as competitors for retrieval (except perhaps in cases where a single phoneme may be analysed as a word in its own right like /aɪ/ as the personal pronoun I). In Section 6 we will take a look at these phonemic cueing techniques in more detail before making some suggestions for their adaption for use with signed language users.

5.2. Phonological priming in signed languages

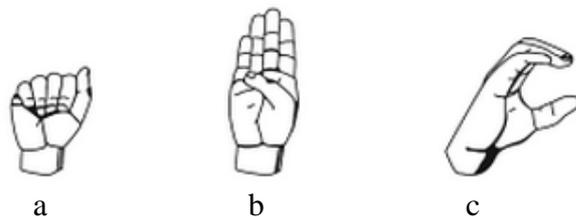
Most theories of phonological priming have been based on studies of spoken languages with relatively few examining how phonological priming takes place in the mental lexicon of signed language users, but this is not very surprising, really, given that until fairly recently most phonological research has concentrated on spoken languages rather than on signed ones.

There is strong evidence to suggest, however, that the same phonological system that underlies spoken languages underlies signed languages as well (Sandler & Lillo-Martin 2006), and if it is the case that their phonological systems are the very same, it should follow that phonological processes occur in each modality in the same way. Parallels were first drawn between the phonologies of spoken and signed languages by Stokoe in 1960 who classified signs according to the shape of the hand, the location of the hand in relation to the body and the

movement of the hand through the signing space. He proposed that signs are constructed from a limited set of formational elements drawn from three articulatory parameters: handshape, movement and location (Stokoe 1960, Stokoe et al. 1965) which are formally analogous to the three phonological parameters of speech production: voicing, place of articulation and manner of articulation (Chomsky & Halle 1968). Just as the voicing, place of articulation and manner of articulation parameters of spoken languages combine to produce phonemes, he said, the handshapes, movements and locations of signed languages come together to create the smaller phonological units of signed language phonology, cheremes (Stokoe 1960).

Some of the most commonly used handshapes in signed languages are FLAT HAND, FIST HAND and CUPPED HAND (Ottenheimer 2012). Rather self explanatorily, in a FLAT handshape the hand is flat, in a FIST handshape the hand is rolled into a fist and in a CUPPED handshape the hand is curved as in Figure 6 in which a FIST handshape, FLAT handshape and a CUPPED handshape are labelled a, b and c:

Figure 6. Some handshapes of a signed language (adapted from Ottenheimer 2012: 123)



Any one of these handshapes can then be combined with a movement (such as UP, DOWN, TOWARD THE BODY, AWAY FROM THE BODY or ACROSS THE BODY) and a location (such as FACE, HEAD or UPPER BODY) to form a chereme, which can together combine with other cheremes to make up a sign.

Psycholinguistic studies in more recent years have gone on to explore whether the phonological priming that occurs in spoken languages can occur in signed languages as well (Carreiras et al. 2007). Corina (2000), for instance, conducted a series of experiments to investigate whether phonological parameter values of signs could be used for priming. Since phonological priming would be indicated by faster response times when a sign was preceded by a phonologically related sign, compared to an unrelated sign, Corina (2000) used a lexical decision task where deaf signers would view two sign stimuli in succession and be asked to decide whether the second sign was a true sign or a nonsign. The sign pairs either shared the same movement, the same location, or were phonologically unrelated (i.e. shared no phonological parameter values). Corina (2000) found no evidence for priming with either movement or location, which contrasted with results from an earlier experiment she conducted with Emmorey in 1993 in which lexical decision tasks revealed facilitatory effects when signs shared the same movement as the stimulus and inhibitory effects for lexical access when they shared the same location. When pairs of signs and stimuli shared a common handshape neither facilitation nor inhibition was found (Corina & Emmorey 1993).

Corina & Hildebrandt (2002) investigated priming in signed languages with the movement and location parameters, but found no evidence to suggest that priming occurred with either of the two. Dye & Shih (2006), on the other hand, have reported data from users of BSL which showed evidence of facilitatory effects for both movement *and* location.

Now, although the results of psycholinguistic studies examining phonological priming with handshape, movement and location in signed languages have been decidedly mixed, I would like to argue that this inconsistency may well be because these studies have only made

use of the loose phonological parameters identified by Stokoe in 1960 and by doing so have not actually accounted for phonological variation within those parameters.

We know from studies such as those of Sandler (1989), van der Hulst (1993), Brentari (1998) and van der Kooij (2002) of the variation within the phonological parameters of signed language. Sandler (1989), for example, pointed out that while handshapes may be flat, curved or fistled, the presence or absence of the fingers and thumb and their individual configurations when they are present can give rise to a number of handshapes that are flat, curved or fistled. Either one, some or all of the fingers may be present in a handshape and the thumb may be either present or absent. By the same token, the extremities that are present may be open (i.e. extended), closed (bent at the joint so that the tip of the finger or thumb makes contact with the palm), curved (extended but bent at the joint nonadjacent to the palm), bent (bent at the base joint but not so much so that the tip of the finger or thumb touches the palm) or even spread (fanned).

It would be naïve to think that signers would be insensitive to such variation. We certainly would not assume that speakers are unaffected by it, would we? Consider the following example from spoken English. The English word *dog* is monosyllabic, made up of the onset /d/ and the rime /ɒ g/. The word-initial phoneme /d/ in the onset is a voiced alveolar stop, so has the voicing parameter value [+ voice], the place of articulation parameter values [+ anterior] and [+ coronal] and the manner of articulation value [- continuant]. If the word *dog* was to act as a target in a phonological priming task, it would make more sense for a word (or non-word) that shares its word-initial phoneme to act as its prime, rather than a word that shares its voicing parameter value. This is because there is only one voiced alveolar stop in the English language, but there are a number of other voiced sounds, including the voiced obstruents [b], [g], [v], [z], [ʒ], [ð] and [dʒ] as well as the sonorants [m], [n], [ŋ], [l], [ɹ], [j], [w], [i], [e], [ɛ], [a], [ɑ], [ɔ], [o] and [u], to name but a few. A methodology that primes with phonemes would be likely to yield more reliable results than one that primes with parameter values as it could control better for variation.

Recall from Section 5.1 that in analogy to the phonemes of spoken words, Stokoe (1960) argued for the existence of cheremes, a discrete set of meaningless units that combine to produce a potentially infinite set of signs (Stokoe 1960). I would like to argue that if psycholinguistic studies on sign processing had been more like the studies on word processing and controlled for variation by selecting signs that share a chereme with the target as prime stimuli rather than signs that share a handshape, movement or location, they would have evinced more positive priming results. We would not expect two people with a bacterial infection to recover at the same rate if one was administered antibiotics and the other placebo pills, so why would we expect two different types of prime to have the same effects? It makes sense to predict that phonological priming will occur equivalently in each modality if the primes that are used in one modality are equivalent to the primes that are used in the other, but surely not if they are not. It would be interesting to see then whether signs that are selected as primes by virtue of them sharing a chereme with the target could facilitate word retrieval (or even whether cheremes alone could), as this could have serious consequences for the treatment of phonological anomia in aphasia.

6. Some suggestions for the treatment of phonological anomia in signed languages

The stimuli used in phonemic cueing tasks for the treatment of phonological anomia in spoken languages tends to be a set of line drawings that are presented to patients to be named on confrontation (Snodgrass & Vanderwart 1980). Patients are given a time limit within which they must name a target and if they are unable to respond or instead respond with an error, their response is recorded as incorrect (Nickels 2002, Greenwood et al. 2010). If a patient fails to

name the target correctly within the given time limit, a phonemic cue is provided by the clinician to prime it, which is usually the initial phoneme of the target (Hickin et al. 2001). Processing of the phonemic cue is said to activate its own underlying phonological representation as well as the phonological representations of all words beginning with it (which includes the target word, of course). Following the cue, the subject is then given another time limit within which they must try the target once more. The phonological representation of the target word is said to remain partially active at this stage, allowing phonological encoding to be accomplished more easily (Schriefers et al. 1990, Meyer & Schriefers 1991). Responses are recorded as incorrect or correct in the same manner as before and the percentage of correctly named items post-treatment is compared with the percentage of words correctly named pre-treatment to measure the treatment's efficacy.

In light of the existence of cheremes, I am keen to suggest that the phonemic approach to intervention for spoken word retrieval could easily be adapted for use with signed language users provided that cheremes can prime signs in the way that phonemes (their spoken language equivalent) can prime words. If this is so, the same methodology could be followed, but rather than clinicians providing patients with a phonemic cue when they have been unable to produce the target, they could offer a cheremic cue instead. The cheremic cue could then prime the target sign by activating its underlying phonological representation, making it more accessible and therefore easier to retrieve from memory.

7. Conclusion

To summarise, aphasia is an impairment of language that follows damage to the language area of the left cerebral hemisphere. Extensive research into aphasia in hearing individuals has identified robust patterns of breakdown across a number of spoken languages. We know from this research that the two most common forms of aphasia include Broca's aphasia (a non-fluent expressive aphasia) and Wernicke's aphasia (a fluent receptive aphasia) but that the symptom most commonly associated with all of the aphasias is anomia, an impairment of language which causes word retrieval difficulties. People with anomia may be unable to access words at all, or produce frequent word errors like semantic paraphasias such as calling a chair a *table* (Howard & Orchard-Lisle 1984, Kay & Ellis 1987, Hillis et al. 1990) or phonological paraphasias such as calling a cat a *tat* (Atkinson et al. 2005).

By contrast, there has been scant research into the impact of aphasia on sign language, but from the few studies that have been conducted we know that many of the patterns described above also occur in signed languages; the non-fluent/fluent dichotomy has been described by Poizner et al. (1987) for example and there have even been individuals reported to have sign finding problems as a symptom of their aphasia (Chiarello et al. 1982, Marshall et al. 2004). There are more specific parallels in the nature of aphasic errors, however. For example, in sign aphasias we observe semantic errors that are caused by semantic anomia where signers sign a semantically related word instead of the target word and phonological errors that are due to phonological anomia which entail signers making use of the wrong phonological parameter(s) or chereme(s) in a sign.

Up until recently, the emphasis of previous studies has been placed on the treatment of semantic anomia in spoken languages (Hickin et al. 2002). Treatment of phonological anomia in spoken languages has been studied too, but to a lesser extent, and treatment of phonological anomia in signed languages has been largely overlooked. Phonological anomia in spoken languages is often treated with phonemic cueing tasks in which phonemic cues are provided to patients to phonologically prime a target word and facilitate its retrieval, since phonological priming has been shown for some time now to improve lexical access (Marslen-Wilson & Welsh 1978, Goldinger et al. 1992, Coltheart et al. 2001, Carreiras et al. 2005).

In previous psycholinguistic studies of sign processing, however, the effects of priming on lexical access have been decidedly mixed. I argued that this may well be because in such studies, distinctions between signs are based on parameter values, and signs are selected as primes by virtue of them sharing the same handshape, movement or location as the target word. This means that variation within each of the parameters is neglected, and it would be naïve to believe that sign processing is insensitive to such variation. In psycholinguistic studies of word processing, however, prime stimuli are selected due to them sharing their word-initial phoneme with the target. Since Stokoe (1960) has argued for the existence of cheremes (the signed equivalent of phonemes), I suggested that if we better control for variation within parameters by selecting signs that share a chereme rather than a phonological parameter value with the target as prime stimuli, results would be more reliable and may well evince the positive priming effects that we see in spoken language studies. That said, I went on to suggest that if this was the case we could very easily adapt the current phonemic cueing tasks used to treat spoken language phonological anomia to make them suitable for signed language users. Rather than providing phonemic cues to improve lexical access, we could provide cheremic cues instead. It would be interesting to see whether this could help signers with phonological anomia with their word retrieval, though additional research to test the success of cheremic priming in signed languages would of course have to be conducted before we could attempt to use it clinically. It is about time deaf people had access to phonological therapy in the way that hearing people do, and given the rise of signed language phonology, there really is no excuse for them not to.

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